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ABSTRACT

Intended to dispel myths concerning epilepsy, the paper discusses the history, symptoms and characteristics, possible causes and current medication approaches to the condition, theoretical assumptions are traced, and a definition explained. Charts depict the location of discharge; seizure patterns and accompanying physical/psychological symptoms; precipitating agents for five types of seizures; and prescriptive information for specific medications including dilantin, mysoline, mesantain, and zaroutin. Implications for educators are addressed. (CL)

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EPILEPSY: AN OVERVIEW FOR THE SPECIAL EDUCATOR

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EPILEPSY: AN OVERVIEW FOR THE SPECIAL EDUCATOR

This paper addresses the history, manifestations, possible causes and current medication regime of epilepsy and is presented to dispel for the special educator some of the mystic which still surrounds the disease.

History:

Epilepsy, the falling sickness, dates back in written record to Mesopotamian culture and was, until very recent times, historically entwined with religious and political belief systems. The first monograph on the "Sacred Disease" is found in the Hippocratic collection (400 B.C.) written by a physician for laymen of that era. The Latin "morbus Maior" designated the major epileptic attack. During this period of history, the disease was defined as an infliction or possession by a higher power, hence the cure had to be of a supernatural nature. Cure depended upon the patient being able to find those powers which might be inflicting the illness and then remember all their personal transgressions which might have offended those powers. The sufferer had to know all the procedures and remedies which could ward off or conquer those supernatural powers. (Temkin, 1971)

Epilepsy, while not the only disease designated as sacred, is the primary one to retain that label through time. The Greek (Epilambanein) to seize, to attach; St. Augustine's (passio caduca) falling evil; the German (Fallendes Siechtum); the Hebrew (choli hopel) share the same connotation. By the 7th century, epileptics were called "lunatics" by the populace, a term not coined by physicians. During the more influential periods of the Christian church, periodic ecstasies, raptures, and prophetic trances were associated with epilepsy. (Temkin, 1971)

A lack of knowledge in the areas of physiology and medicine kept this particular affliction surrounded by mystery and subject to the cures and remedies of the shamon and priest. The disease was considered contagious and/or hereditary. Pregnant females with a history of seizures were buried alive in medieval Scotland. Mohammed, Plato, Socrates, Alexander the Great, and Napoleon are among the famous considered to have been epileptic. (Temkin, 1971)

It remained for John Hughlings Jackson, head of the British National Hospital for Relief and Cure of the Paralyzed and Epileptic, to separate epilepsy from its mystic. Jackson worked from 1861 to 1906 and conceptualized epilepsy as a local discharge occurring in the areas of cerebral gray matter. The spread of this discharging state to neighboring areas accounted for the march of motor manifestations seen in the seizure and formed the basis for a rational explanation of epileptic seizures of all types.

Theoretical Hypothesis:

Centrencephalic theory proposes that seizures are directly generalized to a large part of one if not both hemispheres. Such seizures originate in the brain which is situated in the centrencephalic system and from which diffuse projections channel very rapidly, permiting instantaneous involvement of the whole brain.

Cortical theory suggests that generalized seizures are always secondary to a discharge which is first localized to one point of the cortex, then is rapidly propogated to the whole cortex through the intermediary of various association pathways (intra cortical, sub-cortical, commissural, etc.). An allocortical sub-variety of this theory is held by some who

think that the hippocampus may be the usual 'pacemaker' for generalized epileptic discharges.

Cortico-subcortico-cortical theory holds that generalized seizures depend on a discharge involving at first a very limited part or a larger part of the cortex, but secondarily propagated to the "centrencephalic system" which would be responsible for the secondary generalization.

Holocenphalic theory has not, to the knowledge of the cited authors, been considered as to which generalized seizures would be dependent on a massive discharge of all encephalic neurons, without passing by the intermediary of a centrencephalic or cortical 'pacemaker'. (Gastaut, Jasper, Bancaud, Waltergny, 1969)

Definitions:

The 1977 Merck Manual includes the following statement:

> "Epilepsy: A recurrent paroxysmal disorder of cerebral function characterized by sudden, brief attacks of altered consciousness, motor activity, sensory phenomena, or inappropriate behavior. Convulsive seizures, the most common form of attacks, begin with loss of consciousness and motor control, and tonic or clonic jerking of all extremities, but any recurrent seizure pattern may be termed epilepsy."

According to Harold Geist (1962) present-day writers usually speak of epilepsies, produced by a discoverable disease process, as symptomatic and those not due to some organic brain disease or abnormal somatic condition as idiopathic or genuine epilepsy. Geist offered a general description of the chronological symptoms (antecedent to the actual attack) grouped into three main categories:

1. Prodromal symptoms frequently precede the major attacks or manifest themselves before a series of motor attacks. Their significance is unknown, but are suggestive of some general or local metabolic distur-

bance which is quantitatively accumulative. These symptoms can be manifest several days or only a few hours before the attacks and are referred to as "aura".

2. Paraxysmal prodromata may occur in the form of brief dreamy states or temporary subjective feelings of strangeness. There may be sudden myocloric jerks affecting groups of muscles in one or all extremities. Dysesthesias or a pins and needles sensation and sudden momentary abdominal pain, choking, hiccoughs, or palpitation of the heart may also occur.

3. Continuous prodromata may appear for a day or two preceding an attack in the form of increased irratibility or lethargy. By various indescribable actions or changes in disposition, the patient shows evidence of an impending attack. There is usually a voracious appetite, generalized headache, or pallor. Some people become euphoric or ecstatic preceding an attack.

Penfield (1951) believed that epileptic seizures began an abnormal discharge in the centroencephalic nervous system (pons, midbrain, and basal diencephalon) which radiated to the homologous areas of the two hemispheres. Penfield maintained that in the petit mal seizure, the participation of the cortex was more or less passive; in the grand mal seizure, there was a greater intensity and bombardment of the cortex. This mechanism constituted the seizure, whether due to organisity or other causes. The attacks themselves were described by Penfield as the grand mal, the Jacksonian march, petit mal, or psychic equivalent.

In the grand mal, the patient loses consciousness, the muscles become rigid, respiration is suspended and the person falls unless supported. The face becomes pallid due to suspended respiration and skin color returns only when the movements of the seizure go from rigid (tonic) to

jerking (clonic).

The Jacksonian Motor seizure elicits jerking in the muscles of the face, tongue, mouth, and limbs tending to progress in a Jacksonian March: i.e., the attach may involve at onset only one area of the body but the jerkings progress and continue until the entire body is convulsed.

The petit mal attack has as its predominant feature the loss of consciousness which usually begins and ends abruptly. Sometimes the loss is not complete and the attack consists more of twitching motions of the muscles about the eye, recurring with regular frequency, often many times a day.

Psychic equivalent seizures are dominated by the symptom of amnesia. The seizures are longer lasting than petit mal, lasting from a fraction of a minute to hours or even a day. Simple adversive seizures involve an uncontrollable turning of the head and eyes as though the patient were looking over his shoulder without loss of consciousness. The direction of turn is generally held to be to the side opposite the discharge.

Epileptic Seizure Patterns:

Penfield's 1951 work with epileptic patients suffering from tumor delientated the location of discharge for four levels of seizures: (Chart 1)

Chart 1

1. Focal Cortical Seizures	Focus in cerebral cortex
2. Highest Level Seizures (Idiopathic, Genetic, Essential Epilepsy)	Focus most probably in higher brain stem; i.e., diencephalon, mesencephalon.
3. Myonclonic Seizures	Focus in lower brain stem and spinal cord
4. Cerebral Seizures (cause is extra-cerebral or due to diffuse brain disease)	Unlocalized

Chart 2 presents the phenomena of seizure, accompanying physical/ psychological symptoms and the cranial area(s) affected.

Chart 2

Seizure Patterns. Penfield and Kirstiansen. Charles Thomas Publishers: Springfield, Illinois, 1951.

A. Initial Unconsciousness Primary Major		
Unconscious adversive	unconscious from beginning without intro. sensory area turning of head & eyes & body to opposite side of suspected lesion	convulsive seizures grand mal
Lapse of consciousness		chiefly frontal petit mal 20% temporal
Unc. Masticatory	movement of jaw, tongue, face, throat as in act of swallowing, tasting	
B. Initial Motor Phenomena		
Simple adversive	uncontrollable turning of head, eyes as though looking over shoulder with loss of consciousness	lesion of frontal lobe
Jacksonian motor	jerking in muscles of face, tongue, mouth; limbs tending to a Jacksonian march	cerebral tumor indicated in half of these cases
Vocalization	complex movement of muscles of larynx, pharynx & mouth & respiratory muscles	intermediate frontal
C. Initial Sensory Phenomena		
Somatic sensation	buzzing, cramping	
Visual sensation	visual images revolving or rotating, black specks before eyes	occipital lobe
Auditory sensation Vestibular phenomena	roaring in ears, dizziness, sense of turning or defective equilibrium	temporal gyrus temporal lobe
Olfactory sensation	smell of burning oil	temporal lobe including the uncus
Gustatory sensation	peculiar taste	temporal lesions

D. Initial Autonomic Motor & Sensory Phenomena		
Epigastric & Abdominal	nausea, thirst, sinking sensation in stomach	fissure of Sylvius
Epigastric rising	feeling of choking	Sylvian region
Palpitation	cardia aura	Sylvian region
Thoracic Sensation	sensation of thorax	fissure of Sylvius
Mis. Autonomic Phenomena	slow or accelerated pulse, involuntary urination	
E. Psychical Phenomena		
Hallucination	resembles dreams made up of past personal experiences	temporal tending to occipital & pariental
Perceptual Illusion	alteration of things seen & heard; change in relationship to environment	undersurface of frontal or temporal lobes
Fear	aura of vague alarm or intense terror	fusiform gyrus
Forced thinking	obsessive thoughts	frontal lobe
Aphasia	temporary loss of control of language produces silence	gyrus predominately
Automatism	loss of understanding; makes no new decisions and not open to reason: would continue to drive unheeding of traffic lights	
F. Miscellaneous Phenomena		
General Bodily Sensations	general tumor starting, jerkings, malaise, general hot or cold reported	temporal, parietal or intermediate frontal
Cephalic Sensation	true pain or headache	intermediate frontal group
Yawning	tingling sensation between knees & ankles of both legs	Third ventricle and hypothalamus

Causation:

The most common causations of epilepsy in infants are intra-uterine or birth injuries, excessive sedation of mother, and asphyzia at birth. During the post-natal period, however, trauma, tumor, infections, degenerative vacular charge and metabolic disorders can precipitate the onset of epilepsy.

Chart 3

NATURE OF SEIZURE	PRECIPITATING AGENTS
grand mal	medication: Micoren, Piperazine, convulsant drugs, hypoxia, hypoglycemia
grand mal	withdrawal from medication, barbituates and sedatives: chlorad, paraldehyde
unilateral seizures	electrolytes & water: infant metabolism awry due to excessive dehydration caused by high fever or administration of sodium chloride following dehydration
bilateral	Pyriodoxine ddficiency: diet due to use of artificial milk devoid of pyriodoxine B_6 deficity: T.B. patients treated with drugs which deactivate coenzyme of vitamin B_6 in certain cerebral enzymes
bilateral generalized grand mal	sensory over-stimulation--synchronous; EEG is normal or slower over the entire brain in animals tested both with and without cerebral cortex

A high incidence of head injury in children can cause post-traumatic

epilepsy. According to Walker (1949) this label is often employed for the peace of mind of the layiety rather than for its accuracy, since the stigma of idiopathic epilepsy implies genetic fault. Walter stated that a blow on the head causing only a scalp bruise is not sufficient evidence to establish cerebral injury. Convulsions usually begin within two years of a cerebral injury, if such has actually occurred; and, if a longer latent period exists, the possibility of other etiological factors increases. The general manifestations of post-traumatic epilepsy are the same as those of lesions and idiopathic epilepsy; unless some cranial repair is required the treatment of patient and family follows the same course. Clinical assessment is vital in any case, and attention should be paid to the psychological aspects of the illness as it affects the patient.

Gastaut, Jasper, Bancaud and Waltergyn (1969) defined functional epilepsy as that which results from a functional cerebral disturbance, independent of any cerebral organic alteration, as with tumor. They postulated that the cerebral hyper-excitability is transmitted genetically or accidently acquired most often under the influence of metabolic disturbance.

Penfield (1951) reserved the term epilepsy for those cases in which no brain lesion or cortical focus can be demonstrated; i.e., idiopathic or genetic epilepsy belong to this group. Focal cortical seizures are nearly always associated with a locally abnormal process and the similarity of the observable symptoms between idiopathic epilepsy and focal (tumor) seizure bears examination.

Psychiatric Factors:

Studies have been systematically conducted since the inception of personality testing to attempt to determine a psychological base for idio-

pathic epilepsy and to evaluate the degree of mental stress which accompanies this illness. A 1962 study of Guerrant, Anderson, Fischer, Weinstein, Jaros, and Deskins used the Minnesota Multiphasic Personality Inventory, the Bender Gestalt, the Rorschach and similarly normed projective tests to compare adults with several types of epilepsy with other chronically ill groups from the 'normal' population. The specific hypothesis tested was that "functional psychiatric disturbances are more common in psychomotor epileptics than in persons with other chronic illness". This hypothesis was not confirmed. Although maladaption occurred in the areas of sex, relationships with siblings and authority figures for the epileptic groups, the maladaption could be directly traced to societal attitudes about this affliction.

Such attitudes can be exemplified by L. Pierce Clark, M.D. (1918-34) who stated that an epileptic attack occurs as a final break from tension. He described epileptic patients' personality structures as egocentric, supersensitive, suffering from emotional poverty, rigid with prior histories of seizure in the family, exhibiting a stronger than usual death wish and a pathological attachment to the female parent.

E. E. Plass (1936) offered the theory that seizures in the human may be of a protective nature; the organism has a generalized convulsion which is in and of itself a splendid mechanism for overcoming a carbon dioxide deficit and reducing alkolisis assumed to exist. Regularity of seizure suggested to Plass an accumulation of the form of metabolic inbalance which precludes convulsions and then is temporarily relieved by the actual seizure.

The Merck Manual (1977) takes a less judgemental approach:

"Epilepsy, particularly temporal lobe epilepsy, is of psychiatric interest for four reasons: (1) Many of the ictal phenomena (focal

epileptic attacks or auras) appear as psychiatric symptoms such as anxiety, elation, hallucinations, depersonalization or memory disturbance. (2) Psychomotor attacks are frequently misdiagnosed as criminal acts or hysterical behavior, and the patients are sent for psychiatric opinion. (3) Epileptics may suffer a variety of transient or prolonged psychiatric illnesses. (4) Epileptics encounter social problems similar to those of schizophrenics and other chronically disabled persons."

Merck (1977) further expands upon psychological aspects of epilepsy according to the nature of particular attacks. Patients with temporal lobe epilepsy may experience anxiety and apprehension, depression and even suicidal ideas for hours, days, or weeks before an attack. These patients rarely experience feelings of well-being or elation, and the symptoms preceding the attack vary in level of intensity only to be dispelled by an attack of grand mal. The patient with psychomotor attack may remain capable of normal behavior but appear confused and incomprehensible to the unaware observer. When consciousness is sufficiently impaired the patient falls to the ground but does not convulse.

The condition referred to as epileptic twilight state, or epileptic furor, or epileptic fugue is actually status psychomotor epilepsy and results in clouded consciousness for hours, days, or even weeks. Twilight epilepsy describes delirious behavior, the 'furor' patient is highly irriated and possibly violent while the fugue state results in the patient wandering in a state of automatism with clouded consciousness and little or no recollection of identity and history.

Merck (1977) suggests that perhaps 10% of temporal lobe epileptics experience brief or prolonged psychotic states that are clinically difficult to differentiate from primary schizophrenic disorder, particularly paranoid schizophrenia. This condition appears only after the patient has experienced attacks of epilepsy for a number of years. Personality

changes do occur especially in those patients with longstanding temporal lobe epilepsy or poorly controlled seizures. Among the personality changes described by Merck for these patients are: poverty and slowness of thought and speech; a tendency to become selfish and disinterested in others; paranoid feelings and pseudoreligiosity. Affect, though essentially blunted, may be displayed primarily as anger or irritability. Habits may deteriorate and eventually, cognitive changes of a slowly progressive dementing process occur. Adequate control of seizure in the early stages is the only treatment to either retard or alieviate this condition.

Diagnosis and Medication:

"Epilepsy affects about 2% of the population. Most patients have only one type of seizure; about 30% have two or more types. About 90% experience grand mal seizures, either alone (60%) or in combination with other seizures (30%). Petit mal attacks occur in about 25% (4% alone, 21% in combination). Psychomotor attacks occur in 18% (6% alone, 12% in combination)" (Merck, 1977). The Epilepsy Foundation of America (1973) estimated that at least 4 million people have some form of epilepsy. The National Institute of Neurological Diseases and Stroke (1974) estimated that about 2 million suffer from epilepsy but some authorities consider both estimates too low and believe the incidence may be as high as 6 million. Geist (1962) stated that epilepsy occurs among relatives of clinical patients about five times more frequently than in the general population. The tendency is inherited and, with a given predisposition, almost anything can be the precipitator.

Clinically-induced seizures using Metrazol accompanied by EEG monitoring have further delineated the nature and extent of a patient's

attacks. Observed changes may be quantiatated and the precise temporal sequence analyzed. Chemically-induced seizures are a diagnostic tool of the greatest accuracy and serve as a means of investigating the morphology and propagation of epileptiform discharge within the brain. This procedure is especially important in incidents where surgery is contemplated or when usual medication has been unsuccessful (Admone-Marson and Ralston, 1957).

In the last 100 years, medicine has progressed from treating epileptics with frog blood, dog urine and amulets to a regimen of highly sophisticated medication administered by prescription according to type and intensity of seizure, modified by body weight and individual metabolic needs.

Some commonly prescribed medicines, their properties and side effects are considered here in two general categories; those medications prescribed for patients with grand mal and Jacksonian epilepsy; and, medication given to those patients with petit mal. This information is included here to re-emphasize the magnitude and variety of epileptic seizure and stress the importance of accurate diagnoses and medical regimen. This list was selected in consultation with local physicians. There seems, to this author, to be two major age classifications regarding epilepsy; the neonate and the adult. The magnitude of symptoms and behaviors in adults parallels the affliction as it relates to children, given the language and experiential lag between the two. We are forced to conclude that symptoms and location of discharge are sufficiently similiar so that studies of adult epileptics can assist us in understanding the epileptic child.

Source: Physicians Desk Reference (1979)

Grand Mal Seizures:

Medication:	Mebaral (Mephobarbital)
Indications for use:	As an anticonvulsant, for the treatment of epilepsy in grand and petit mal.
Countraindications:	Hypersensitivity to barbituates
Warnings:	Tolerance, psychological dependence and physical dependency may occur. Complete list page 689 PDR, 1979.
Precautions:	Caution and careful adjustment of dosage used with patients with impaired cardiac, hepatic, renal or respiratory function. Complete statement page 690 PDR, 1979.
Adverse Reactions:	CNS effects: dizziness, headache, "hangover" confusion, paradoxical excitation, exacerbation of existing pain, nausea, vomiting, hypotension, facial edema, skin rash. Severe allergic reactions may result in degenerative changes in the liver, anemia, thrombocytopenia. Complete list page 690 PDR, 1979.
Medication:	Dilantin Kapseals Dilantin (Parenteral; i.e., injections)
Indications for use:	Oral: for the control of grand mal and psychomotor seizures. Parenteral: for the control of status epilepticus of the grand mal type and for prevention and treatment of seizures occuring during neurosurgery.
Countraindications:	History of hypersensitivity to hydantoin products, both oral and injection types. Parenteral contraindicated in patients with Adams-Stokes syndrome, sino-artrial block, sinus bradycardia.
Warnings:	Abrupt withdrawal of Dilantin and similar products from the epileptic patient may precipitate status epilepticus. Pages 1282-83 PDR, 1979, contains the complete warnings section regarding use of oral and parenteral Dilantin.

Precautions:	Include signs of toxicitin in the gravely ill or elderly, lymph node enlargement. Page 1283 PDR, 1979, specified total precautions list for this drug in both forms.
Adverse Reactions:	Nystagmus, ataxia, slurred speech and mental confusion. Dizziness, insomnia, transcient nervousness, motor twitchings and headache have been observed. A complete list of adverse reactions may be found on page 1283 PDR, 1979.
Medication:	Mysoline (brand of primidone)
Indications for use:	In the control of grand mal, psychomotor and focal epileptic seizures.
Contraindications:	Patients with porphyria and those hypersensitive to phenobarbital.
Warnings:	Abrupt withdrawal may precipitate status epilepticus. Efficacy of dosage regimen takes several weeks to assess. Page 612 PDR, 1979.
Precautions:	Complete blood count and a sequential multiple analysis-12 (SMA-12) should be made every six months. Page 612 PDR, 1979 lists further precautions.
Adverse Reactions:	Ataxia and vertigo are common. Nausea, anarexia, vomiting, fatigue hyperirritability, emotional disturbances, sexual impotence, diplopia, drowsiness and skin eruptions have been reported. See PDR, 1979, page 612 for further information.
Medication:	Mesantain (mephenytain tablets)
Indications for use:	Control of grand mal, focal, Jacksonian and psychomotor seizures in patients who have been refractory to less toxic anticonvulsants.
Contraindications:	Hypersensitivity to hyduntain products
Warnings:	Used only after safer anticonvulsants have been given adequate trial and have failed. Page 1507 PDR, 1979.
Precautions:	Patient must be kept under close supervision at all times since serious adverse reactions may emerge. Read further page 1507 PDR, 1979.
Adverse Reactions:	See page 1507 PDR, 1979.

Medication:	Valium Tablets (diazepam)
Indications for use:	Status Epilepticus and Severe Recurrent Convulsive seizures.
Contraindications:	Hypersensitivity to the drug and in children under 6 months of age. Page 1468 PDR, 1979.
Warnings:	Patients should be warned against engaging in hazardous occupations requiring complete mental alertness. Abrupt withdrawal associated with temporary increase and severity of seizures: Psychological/Physical dependence. Page 1468 PDR, 1979.
Precautions:	Used in combination with barbituates, phemothiagines, narcotics, Valium may become potentiated. Complete section of precautions Page 1468 PDR, 1979.
Adverse Reactions:	Drowsiness, fatigue, ataxia. Sometimes associated with confusion, constipation, hypotension, incontinence, jaundice, changes in libido. Page 1469 PDR, 1979.

Petit Mal Seizures:

Medication:	Paradoine (paramethadoine)
Indications for use:	For the control of petit mal seizures that are refractory to treatment with other drugs.
Contraindications:	For patients with a known hypersensitivity to the drug.
Warnings:	Because of its potential to produce fetal malformations and serious side effects, paradione should only be utilized when other less toxic drugs have been found ineffective in controlling petit mal seizures. May cause serious side effects. STRICT medical supervision is mandatory, especially during the initial year of therapy. See page 550 PDR, 1979.
Precautions:	Abrupt discontinuation may precipitate petit mal status. See Page 551 PDR, 1979.
Adverse Reactions:	Not all of the following side effects have been reported with the use of Paradione but are associated with oxazolidinedione compounds to which group Paradione belongs.

	Nausea, vomiting, abdominal pain, gastric distress, drowsiness, fatigue, malaise, insomnia, vertigo, headache, parathesias, precipitation of grand mal seizures, personality changes, increased irritability, bleeding gums, retinal and petechial hemorrhages, hypoplastic anemia and fatal aplastic anemia. Skin rash, hiccups, anorexia, weight loss, hair loss, changes in blood pressure, albuminuria, fatal nephrosis has occurred and rare reports of hepatitis. Complete lists and further explainations page 551 PDR, 1979.
Medication:	Celontin (methsuximide capsules)
Indications for use:	Control of absence (petit mal) seizures that are refractory to other drugs.
Contraindications:	Patients with a history of hypersensitivity to succinimides.
Warnings:	Blood dyscrasias, including some with fatal outcome. Periodic blood counts mandatory. Use care when administered to patients with known liver or renal diseases. Page 1277 PDR, 1979. Methsuximide may impair mental and/or physical abilities required for tasks such as driving a motor vehicle.
Precautions:	Gradual withdrawal recommended on appearance of unusual depression, aggressiveness or other behavioral alterations. See page 1277 PDR, 1979.
Adverse Reactions:	Nausea or vomiting, diarrhea, weight loss, epigastric and abdominal pain and constipation. Complete section on page 1277 PDR, 1979.
Medication:	Zarontin (ethosuximide, USP)
Indications for use:	Control of absence (petit mal) epilepsy
Contraindication:	Patients with history of hypersensitivity to succinimides.
Warnings:	Blood Dyscrasias, including some with fatal outcome have been reported. Periodic blood counts mandatory. In humans, abnormal liver and renal function studies have been reported. Page 1326 PDR, 1979.

Precautions:	When used alone in mixed types of epilepsy may increase the frequency of grand mal seizures in some patients. Abrupt withdrawal of anticonvulsant medication may precipitate petit mal status. Page 1326 PDR, 1979.
Adverse Reactions:	Anorexia, vague gastric upset, nausea and vomiting cramps, epigastric and abdominal pain, weight loss and diarrhea. Leukopenia, aplastic anemia, drowsiness, headache, dizziness, euphoria, hiccups, irritability, hyperactivity, lethargy, fatigue and ataxia. Psychiatric or psychological aberrations including disturbances of sleep, night terror, inability to concentrate, aggressiveness. Drug effects seem to aggrevate pre-existing psychological abnormalities. Complete section on page 1326, PDR, 1979.

Epilepsy--Prognosis:

"Drug therapy can control grand mal seizures in 50% of cases and greatly reduce the frequency of seizures in another 35%; control petit mal seizures in 33% and reduce the frequency in 33%; control psychomotor attacks in 28% and reduce the frequency in 50%.

Most patients with epilepsy are normal between attacks, although overuse of anticonvulsants can dull alertness. Progressive mental deterioration is usually related to accompanying organic brain damage although it can also occur with repeated uncontrolled seizures. The outlook is better when no brain lesion is demonstrable. About 70% of noninstitutionalized patients with epilepsy are mentally normal, 20% show a slight reduction in intellect, and 10% have a moderate to pronounced impairment." (Merck 197

Summary:

Epilepsy is sufficiently common in schoolaged children to merit the

attention of special educators. This paper attempts to bridge the gaps between myth and fact in the areas of history, causation, diagnosis and treatment.

There is every indication, especially concerning metabolic-based seizures, that prevention in the pre-natal period may radically diminish the number and intensity of epileptic seizures in children. Vitamin deficiencies due to improper diet and accidental abuse of medication in the pregnant mother may account for much of the birth and post-natal malfunctions. Better attention to general health in mother and infant can further reduce the incidence of epilepsy as a result of dehydration trauma. The complexity of the disease is further attested to by the multiple side-effects and risks which must be borne by the patient with convulsive tendency. Educators are cautioned further about making medical referrals of children suspected of being epileptic since the cure, when inadvisedly administered, can be as harmful as the application.

Teacher training programs should prepare student teachers to observe and adequately record those behaviors in children which may be associated with epilepsy. University programs should include emergency first aid training to enable teachers to respond appropriately in emergencies.

It behooves educators to establish an especially strong rapport with parents so that school behavior and observable drug reaction can be shared. Without an on going parent/teacher interaction from the time of referral through diagnosis and throughout treatment the child's attacks may be inadequately controlled.

While serious cranial trauma often results from true accidents, educators can be alert to post-natal injuries inflicted by battering

parents. Dr. David Hefelfinger, chairman of the University of Alabama Department of Pediatrics (1980), has noted that 300,000 cases of child abuse are reported annually in the United States. Dr. Hefelfinger suggests that for each reported case as many as 10 go unreported and untreated. Among this staggering number of abuses will be children whose cranial trauma is serious enough to precipitate idiopathic epilepsy so long as two years beyond the injury.

The Physician's Desk References serve as a reminder to educators that epilepsy is continual hence the behavior of epileptic children must be constantly monitored for the possibility of adversive reactions to the medical regimen.

Local Public Health departments and The National Foundation for Epilepsy provide updated information in the form of brochures which instruct the general public on the nature and treatment of this disorder. The better educated parents and teachers are regarding diseases such as epilepsy, the more likely it is that epileptic children can be helped to live normal lives.

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This book cites the work of Penfield and Dristiansen over a 10 year period with 259 patients treated in the Montreal Neurological Institute. The graphic representations of lesion location and the definitiveness of various forms of epileptic aura are especially helpful. Penfield is considered a leading authority on epilepsy.

Physician's Desk Reference, 1979 Edition.

Provides lists of drugs, their action and uses, quantity of dosage and precautions to the user.

Strauss, Kephart, Brain-Injured Child, Vol. II, Progress in Theory and Clinic, Grune & Stratton, New York, 1955.

This text was chosen for the clarity of cerebral diagrams. The section of essays on mental functioning of brain injured children and the chapter on testing are quite helpful.

Temkin, Owsei, The Falling Sickness, Sec. Ed., John Hopkins Press, Baltimore, 1971.

A completely footnoted historical overview of epilepsy from antiquity to modern day. Each page is half text and half footnotes with a wealth of resources for the reader. The author restricts himself to the concept of epilepsy in the Western World and the book contains an overview of political and religious practices valuable beyond the topic of epilepsy.

Walker, Earl, Post-traumatic Epilepsy, Charles Thomas Publisher, Springfield, Illinois, 1949.

This monograph deals at an elementary level with diagnosis and treatment of epilepsy symptomatic of cranial injury.